Oxygen and Its Pivotal Role in Immunomodulation

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Introduction

By carefully studying the immunomodulatory effects of tea tree oil upon autoimmune conditions such as psoriatic scalp, insights can be gained into the role of oxygen in modulating autoimmune conditions and in localized tissue hypoxia in aggravating the same conditions.

Abstract

I propose that that tea tree oil has as one of its properties the ability to bind to atmospheric oxygen and is therefore capable of carrying oxygen deep into the dermus. I propose that it is the profusion of oxygen deep into the scalp which provides the immunomodulatory effects associated with tea tree oil for those suffering from psoriatic scalp. Incredibly, this highly-effective remedy for psoriatic scalp is better known to beauticians than it is to doctors and no studies have been carried out into its mode of effication. If one simply studied this substance vis-à-vis psoriasis, he or she would gain an important insight into the human immune system.

Billions of dollars are being invested into research into immunomodulator drugs meant to treat a wide variety of autoimmune conditions. Although not all of them can be treated with a medicated shampoo, this research could be greatly simplified and accelerated through the understanding that *most autoimmune conditions are triggered by transient or chronic localized tissue hypoxia*. There is an evolutionary basis for this conjecture.

When a patient presents with a dermal infection with an aerobic bacteria, one of the consequences of such infections is that the bacteria will consume oxygen, causing localized tissue hypoxia. The human immune system is generally able to attack the bacteria and usually entirely eliminate it, however, the immune system needs a quick and simple means of knowing when it needs to send additional white blood cells to a specific area. If a person is infected with a novel strain of bacteria to which he or she has no immunity and that immunity requires time (days, perhaps) to develop, how then, is it, that the immune system is able to react to the infection and for puss to form around the infection site? I propose that T-cells are sensitive to local levels of tissue oxygenation and that this sensitivity is leveraged to trigger an immune response to dermal infections on the basis of the detection of localized tissue hypoxia. This is a response which is likely shared in common with other species and has evolved over a great deal of time. If transient localized hypoxia can trigger a short-term immune response, it stands to reason that such reactions are switchable and that, sometimes, such reactions never cease due to a failure of the immune system to switch back to their original settings, or because the immune system detects the presence of some additional substance which is associated with an established antigen (as in an allergic reaction.)

If this is the case, it seems likely that the opposite would also be true. If we were to introduce oxygen to localized areas by, for example, saturating tea-tree compounds with large concentrations of oxygen prior to dermal application, it would have a clear immunomodulatory effect, perhaps suitable for treating conditions such as eczema. For example, a salve which is marketed and packaged along with a small canister of liquid oxygen could include instructions to inject a small quantity of the liquid oxygen into a pre-measured portion of the salve and to vigorously mix the two prior to applying to skin. This unusually concentrated profusion of oxygen to the dermus in a localized area could break the cycle of dermal autoimmune reactions without the use of steroids and with a greater degree of efficacy and without the side-effects associated with topical steroidal ointments.

Targeted Oxygen-Mediated Immunomodulation

I propose that the goal of designers of immunomodulator drugs should be to identify the molecule which is acting as an antigen in the immune reaction and to introduce oxygen into the immediate area of that antigen. If we take the example of a largely discontinued antibacterial substance called triclosan, it was found that triclosan would actually cause patients to become allergic to substances to which they had not previously been allergic if they ingested small quantities of the trioclosan along with the other substance (gluten, peanuts, et cetera.) At the essence of anaphylaxis is a phenomenon in which the immune system attacks virtually the entirety of the human body, resulting in the global release of cytokines and the associated symptoms of airways closing off, the constriction of blood vessels, tissue inflammation. et cetera.

Triclosan perniciously bestows upon patients novel allergies by binding to dietary molecules such as gluten or peanut oil and by persisting in the body in that bound state until the triclosan eventually succeeds in disrupting fatty acid synthesis during a human cell replication event, somewhere in the body. Triclosan's mode of effication in killing bacteria is known to be fatty acid synthesis disruption. It can therefore be inferred that it is capable of doing the same thing to human cell (membranes) during cellular replication. When this happens, the immune system treats the cells as antigens but also begins to treat the food ingredient as an antigen and it remembers the association between the two. When triclosan-induced allergy is introduced, the immune system begins habitually attacking the entirety of the body (or specific groups of tissues) any time the immune system detects the food ingredient in the future.

Triclosan is not the only substance capable of both disrupting cell formation and binding to food molecules. Allergies have as their root cause exposure to substances which happened to be present in the same part of the body where cell apoptosis needed to be triggered at that same time, for any reason. It is well-known that the severity of allergic reactions can be reduced by purposefully introducing a patent to increasing quantities of the substance to which they are allergic. I don't think it an adequate explanation to say that the immune system "gets tired of" attacking the body after so many exposures to small quantities of the substance, e.g. peanuts and yet, this is the explanation we have been given.

I propose that what is actually happening when a patient is treated via Allergen Immunotherapy is that the same sort of chance collocation of immune cells and allergens which gave rise to the allergy in the first place (e.g. gluten plus triclosan plus cell apoptosis caused by the triclosan,) with the key difference being that instead of something harmful like triclosan mediating the event, a free oxygen molecule is present and serves as an immunomodulator, resulting in a reprogramming of the immune system. When T-cells detect an oxygen molecule, they automatically treat this as a "safe" condition and any molecule which happens to be in the presence of the T-cell at the time is reported as "safe," by association, even if it had at one time been treated as an antigen or allergen. I propose that T-cells behave in this way on the basis of evolution. As we are aerobic creatures, our own natural tissues are inherently oxygenated and, therefore, anything which is oxygenated is inherently "good," from the perspective of the T-cells. This behavior of our immune system has been crucial to our survival not only in the case of infections, but for helping to survive tissue necrosis, given that the immune system needs to know how to sequester the dead tissue. I propose that this function is also governed by an ability of the cells to detect oxygen.

Taking all of this into consideration, I propose that it would be reasonable to isolate antigens and to introduce them into the body in large quantities with the caveat that they be bound to oxygen molecules prior to introduction. It should be noted that a true molecular bond may not be necessary as the antigen-oxygen combination might also be encapsulated in lipid sphere which releases both an antigen and an oxygen simultaneously. Physical collocation is what is needed in order for the immune system to associate the antigen with healthy tissue once again and for a beneficial immune reprogramming to occur. This may explain the mixed results experienced in Allergen Immunotherapy and recommend a mechanism for ensuring its greater efficacy.

Conclusion

By using the observed immunomodulatory effects of tea tree oil as a starting point, I propose that the preceding series of straightforward inferences about the human immune system may be confirmed in future studies, leading ultimately to highly effective treatments for a wide range of autoimmune conditions.